A convenient laboratory method for the determination of the thyroid content of the blood, just as hemoglobin is estimated, is needed very much in medicine.

To Dr. Walter H. Andrus, of Germantown, Philadelphia, the writer is indebted for notes of two cases and for the privilege of seeing one of them. To Dr. Harry Harvey also the writer owes his indebtedness for notes, etc., in one case seen with him.

FIVE YEARS' EXPERIENCE IN THE TREATMENT OF PULMONARY TUBERCULOSIS BY AN ARTIFICIAL PNEUMOTHORAX.[1]

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In 1909 the Highlands Camp Sanatorium began the treatment of pulmonary tuberculosis by an artificial pneumothorax according to the principles established by Forlanini, the author and perfector of the method.

The most important points for us have been the determination of the effect of the method upon the contralateral lesions and the decision as to whether these lesions indicate or contra-indicate the method; the pros and cons of delay; the quantity and frequency of the fillings; the amount of pressure; how best to break up adhesions, stop hemorrhages, prevent and treat pleural effusions.

In determining whether the contralateral lesions permit or forbid an artificial pneumothorax, we have to consider whether the condition of the patient is serious enough to warrant the attempt as an *ultimum refugium;* the extent and the nature of the lesions; the probable effect of suppressing the absorption of toxins; the probable effect of increased intrathoracic pressure upon the better lung and interference with its circulation.

We have had two cases come to us as a last resort, perfectly aware that failure and death were far more probable than recovery; both lived for more than two years, possibly because the absorption of toxins from the bad lung was suppressed. Another case with superficial, fine, wet rales all over the better lung in front from the second to the sixth rib; with absence of first sound of the heart and no palpatory apex beat; soft, irregular pulse and decided tendency to intermission; with the upper lobe of the bad, right lung almost entirely excavated, and the rest of the lung full of degenerating foci, offered little hope of recovery from an illness of more than two years in bed. There was marked cachexia, total loss of appetite,

[1] Read at the Meeting of the Tri-State Medical Association, Charleston, S. C.

periodical attacks of temperature of 101° F. The right lung and cavity was compressed without any difficulty; the rales in the left lung disappeared and the heart action improved. The patient is out of bed, comes up quite a hill for his fillings, and promises a recovery.

Another case of extensive rales in the better left lung with similar cardiac conditions was regarded as hopeless. Again, the compression of the cavities in the bad lung was followed by the disappearance of rales in the other and by improved heart action. This patient is earning his living. Neither the physical signs nor the Roentgen-ray encouraged us to hope for results in these four cases, and it was only as a last chance that they were undertaken.

In the last two cases it is possible that the wet rales were caused by the dilating influence of the toxins, and that this was comparable to an excessive dose of tuberculin upon vessel walls injured in their tonicity by the long-continued action of poisons. The Roentgen-ray showed extensive peribronchitic infiltrations of these left lungs together with old scars, but no unmistakable evidences of activity. In these cases the rales indicated leakage through vessel walls rendered more permeable by tuberculins and not degenerative processes. We have had several bilateral cases similarly benefited, so that the extent of wet sounds in the better lung does not seem to us as prohibitive as it once did, provided the Roentgen-ray suggests absence of active lesions. On the other hand, we have had several cases of absence of rales in the better lung, and apparently only scars revealed by the Roentgen-ray, in which the increased functional demands upon the lung or the increase in intrathoracic pressure caused the appearance of rales in the region of the heart, which in spite of all precautions increased and affected the integrity of the lung. In one of these cases there had never been any abnormal breath sounds or rales in the better lung, and the compression of the bad lung had caused the disappearance of the clinical signs of tuberculosis, when a severe nervous shock brought on an edematous infiltration of the better lung, from which the patient never recovered.

If the contralateral lesions are severe it is possible that delay will increase them so that the attempt can never be made and that prompt action is the one chance. We are often tempted to delay, hoping that the lesions will improve and give us a better chance for holding out against the increased strain. We waited for this improvement recently, but by so doing lost the opportunity. We are not able to predict whether these serious cases will be improved or harmed by delay, and it is only the outcome which tells us whether our judgment has been right or wrong. A case of 103° F. temperature with almost daily chills, aching, and other signs of toxemia improved after resting in bed for a year, so that we were not afraid to undertake the compression of the thoroughly bad lung.

The better lung has steadily improved and the prospects are good. Possibly if we had begun sooner the lung could not have stood it. Several times delay has seemed to assure recovery by getting the lung in much better condition, so that a case which was contra-indicated became possible.

In two cases we should have begun the compression a year before we did. In one there was caseous pneumonia of the right upper lobe which we thought would heal without any interference. During the fall and winter the lung was dry and apparently cicatrizing. There was every indication of satisfactory recovery until the warm weather came, and then rales reappeared and a little blood was coughed up, so that it seemed best to compress a lung that broke down so easily. A good recovery was obtained with working capacity, which persists, but if we had compressed the lung when the patient first came to us we would have saved him nine months' time, all the expense, and loss of working capacity, and the consequent discouragement and dissatisfaction.

A second case was that of a small cavity in the lower part of the right upper lobe, which we were confident would heal. The patient gained in every way for eight months, and then severe hemorrhages induced us to compress the cavity. Our mistake is very evident looking back, but we had just begun the work, and our first principle was never to use the method unless compelled to, and so we cost this patient his time and money and a strong sense of impatience and loss of hope. Another case was wiser than we. Told that compression afforded greatest security against loss of time and toward recovery, he preferred taking the chances incident to dangers and be on the safe side, although the chances were in favor of a prompt recovery without the need of interference.

Murphy, in his unbounded enthusiasm, urges the treatment of early cases regardless of the possibility of recovery without it, and compares the condition to appendicitis with all its danger of delay. Forlanini also advises its use earlier than is commonly deemed advisable.

As we learn the ease and certainty of recovery in uncomplicated cases, it seems more and more as if the risks of delay outweighed those of prompt action. If some public sanatorium would furnish statistics from a large enough number of cases treated before the necessity for the method was apparent, we could judge better whether to still advise delay until sufficient harm was done to warrant our interference, or forestall delay and increase the ease of technique by prompt action. We are in favor of regarding earlier cases as indicating the method and of giving it a much wider field than we have hitherto.

For a time we tried to determine the result of a filling by the area of tympany and the absence of breath sounds, together with metallic clinking or breathing and the coin tests. We find that the

area of the pneumothorax can be roughly mapped out by these means, but that the condition of the lung is not revealed. With all the physical signs of a complete pneumothorax the Roetgen-ray may show the sounder portions of the lung well compressed while dense infiltrations remain prominently exposed and uncompressed. Cavities that give no sign are seen to be surrounded by adhesions, and the use of screen or radiographs furnishes the only reliable information. We try to give as few fillings as possible and to use as little pressure. The complete pneumothorax may easily depress the diaphragm, causing insomnia, loss of weight, extreme nervousness, and rales in the other lung. The pressure may be low, even below that of the atmosphere, and yet the diaphragm be too much depressed. If adhesions hold the heart one way and the gas force it another the action is embarrassed. A weak mediastinal pleura may not register any pressure, but nevertheless, the Roentgen-ray shows it forced over until the other lung or the heart is embarrassed. Deep mediastinal pressure may fatally affect the action of the heart if it is a flabby muscle and the danger not be shown by the manometer. The dyspnea is not necessarily great, nor any other marked discomfort. The distress may be vague and ill defined, and the patient suffer more from inability to sleep than anything else. There is nothing in the physical signs to explain the danger, and only the Roentgen-ray shows it. The readings of the manometer are inaccurate guides when the mediastinal pleura stretches and yields instead of raising the pressure.

The breaking up of adhesions seemingly demands higher pressure, but it is possible that too sudden a rise in pressure may account for many cases of pleural effusions, empyemas, perforations, and spontaneous pneumothorax. In judging of a series of cases like Vollhard's it would be interesting to know the pressure and the Roentgen-ray picture. It is safer to give adhesions time to thin out and not to attempt to stretch them too rapidly. If the thick bands are too resistant, or the synechiæ around cavities are too dense to separate, cavities may have to be compressed by either breaking up the adhesions by pneumolysis or a thoracoplasty tried. Some very discouraging cases of adhesions with uncompressed portions of the lungs containing cavities have finally yielded to low pressure, persistently maintained by the frequent giving of small quantities of nitrogen without causing enough pull on the surface of the lung to cause a pleurisy or perforation. The same is true of cavities with bleeding vessels. In hemorrhage cases it is necessary to watch the compression of the lung with a Roentgen-ray in order to see whether the vessels in the thick walls of a cavity may not be subjected to a strain which will cause hemorrhage when the rise of pressure cannot compress the cavity but simply changes its shape. Although severe cases of progressive hemorrhages demand severe compression, it is not always safe to use high press-

ure, because the cavity may bleed and also because the pressure from below may increase the congestion of the uncompressed portions and favor hemorrhage. Some hemorrhage cases do splendidly for a certain time, and just as all seems to be over and the danger-point passed, a severe hemorrhage may be aspirated into the other lung and cause death. Even a very small cavity surrounded by compressed tissues may torment the patient by the frequent spitting of streaked sputum. Even when there has been dangerous hemorrhages, and we are anxious there shall be no more, high pressure should be avoided unless the Roentgen-ray shows safety. Again, the readings of the manometer may be misleading by reason of the stretching of the mediastinal pleura and a severe hemorrhage first inform us that a deep mediastinal pressure has altered the shape of the cavity and started bleeding. A case of persistent hemorrhages for six years with advanced bilateral lesions, bled so profusely that he was nearly strangled. Half-fainting and all unconscious of our efforts, he was held up from behind in a sitting position while 800 c.c. of oxygen were given. Oxygen was used because he was so livid and lifeless that we were afraid the heart could not stand the strain, and also because we hoped it would have a stimulating effect by being absorbed. The color came back, the patient revived, and all was well until nitrogen was used, because the rapid absorption of the oxygen did not keep up a steady pressure. Eight hundred c.c. of nitrogen were given and an adhesion pulled out the surface of the lung, a tension penumothorax developed, and he died. If oxygen had been used and the pressure raised to +2, the pressure used with the nitrogen, it is quite possible that the increased pressure would have forced a more rapid absorption of oxygen and taken the strain off the adhesion, and there would have been no perforation. We are confident that when the pressure is raised with adhesions, oxygen is safer than nitrogen.

In another hemorrhage case lasting for three days, and frightfully severe, we could get no readings with the manometer. We argued that the lung was stiff and rigid with hemorrhagic infiltrations, and could not be affected by the respiratory movement of the chest wall. We were anxious to prevent any more bleeding by a thorough compression, and considered a Brauer incision, but waited, hoping that the lung would dry out and give respiratory excursions. After two weeks the breath sounds began to be heard faintly over the dull areas, and in a few days more there was no difficulty in obtaining readings on the manometer. The lung was completely compressed without difficulty, and the patient is making a good recovery.

The prevention of pleural effusions depends upon technique and good antisepsis, but even with the best technique it is theoretically impossible, even with a Kjer-Petersen needle, to avoid the carrying in of infection from the deeper layers of the skin. Children coming home from school and filling the house with "colds" or

grippe, or bronchitis, or any other infection, are a good cause of pleurisy from acute infections. Chilling, overphysical strain, overeating, and "bilious" condition, etc., predispose. Infectious pleurisies are common after any acute infection. Static pleural effusions form as the result of too much strain upon the injured vessels; the separation of the pleural surfaces favors the dissemination of tuberculous processes over them. The rupture of a tuberculous focus in the periphery, the forcing of such a focus to the periphery by the collapse of the surrounding tissues; the strain upon adhesions and irritation of the adjacent tissues; the rupture of lung substance and formation of a pulmonary fistula from a cavity to the surface; the pulling out of adhesions and consequent empyemas, are all the result of too much pressure, too hastily applied.

Static and infectious pleurisies demand no treatment beyond that of ordinary pleurisies unless there is too much pressure. As a rule, the pressure rises with an effusion, but occasionally it falls even when the fluid reaches as high as the clavicle. Unless there is some symptomatic indication, rest in bed and the ordinary treatment of a pleurisy suffices; but if the dyspnea becomes marked, or insomnia, or the heart action is wrong, or there are vague sensations of malaise that the patient cannot well define, it is well to remember that an effusion can cause just the same deep mediastinal distress as nitrogen, and that this will not be revealed by the manometer but demands the Roentgen-ray. The effect of the fluid is usually good, sometimes surprisingly so, but there are three conditions that need watching: the expansion of the collapsed lung under the fluid, the penetrating tendency of the fluid and consequent danger of perforation, the conversion of the whole pleural cavity into a cold abscess.

Static pleurisies do not have temperature; infectious pleurisies may have a high temperature and stormy course but they are self-limited; tuberculous pleurisies frequently recover, especially when there is a good eosinophilia; the persistent high temperatures with little remission, with night sweats, and general malaise, suggest a cold abscess which will never be overcome so long as the pleural cavity is a box-like structure. These septic pleurisies can only be overcome by a thoracoplasty.

A pneumolysis may suffice for simply an uncompressed portion of the lung. A cavity held out by adhesions may be thus collapsed; it is possible that a compression of the whole lung may not be needed in order to obtain the obliteration of a circumscribed cavity; when an artificial pneumothorax fails it is quite clearly indicated that the uncompressed upper lobe shall be freed by pneumolysis and compressed. If there is a pleural effusion there is danger of empyema. Cold abscess of the pleural cavity does not seem to be within the reach of a pneumolysis, but Sauerbruch has obtained good results by his method of thoracoplasty. Our five years'